

- Cardiology (290)
- Clinical haematology/oncology (171)
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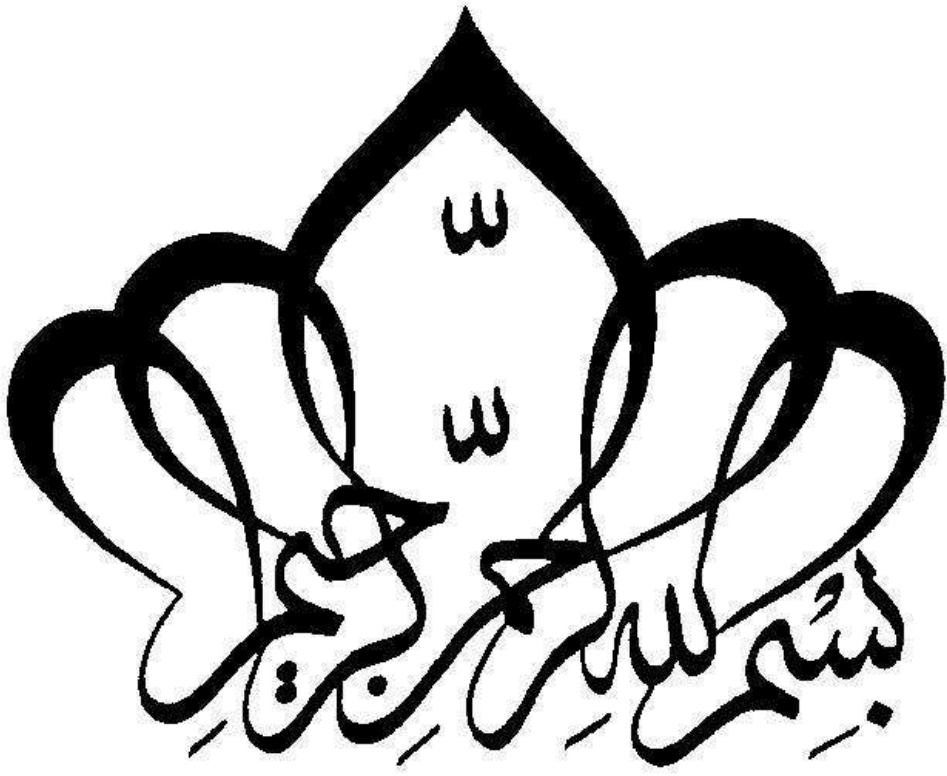
2600 MCQ with Illustrated Answer
Full Topics Note
14 Chapter

Respiratory medicine

P. M MCQ Bank 2017

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قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
۞ إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

إهداء

للكل آية من كتاب الله حفظناها ولكل معلومة قرأناها ونسيناها لعل الله يحمينا من النسيان ويروها إلينا
للكل شخص وعونا له في ظهرك الغيب لعل الله أن يستجيب دعائنا
للكل مريض قرأ له ما أله لعل الله يشفيه ويعير إليه صحته وعافيته
للكل لحظة تألم بها أحببنا لعل الله لا يريهم الألم ولا يضييهم مرة أخرى
للكل موقف اجتجت به عيناى فلم تلبيانى لعل الله لا يضرنى بهما في حياتي
للكل حاتم صاحب أهراف ومباوى لعل الله يحقق له مقاصده حتى يرضيه ويرضى عنه
للكل مجتهد لعل الله يجعل له من التوفيق أوفر حظ ونصيب ويهون له الأسباب كلها من حيث لا يدري من غير ضراء مضرة ولا فتنة مضلة
للكل من فقر شيئا في الحياة لعل الله يعيره إليه أو يعوضه بما يسعده
للكل شخص ابتعدنا عنه لعل الله يجمعه بمن هم أفضل منا
وأخيرا وليس آخرا لأهلنا لعل الله يجعلها لهم صرقة جارية ترو إليهم قليلا من فضلهم علينا وتكون سببا يلم شملنا تحت سقف واحد بعد أن
هجرتنا الحروب والمصائب

يقول أحد الصالحين

إِذَا رَأَيْتَ أَنَّ اللَّهَ وَفَقَكَ لِإِخْوَةِ تَعِينِكَ عَلَى الْخَيْرِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ بِكَ خَيْرًا !! وَإِذَا وَفَقَكَ لِلدُّعَاءِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ أَنْ يُعْطِيَكَ !! وَإِذَا
وَفَقَكَ لِقَضَاءِ حَوَائِجِ النَّاسِ فَأَعْلَمْ أَنَّ لَكَ يَتَخَلَّى عَنْكَ !! وَإِذَا وَفَقَكَ لِلزُّكْرِ.. فَأَعْلَمْ أَنَّكَ مُحِبٌّ !!.. وَإِذَا أَحْبَبَّكَ أَحَدٌ.. وَنَصَرَكَ.. وَأَيَّرَكَ..
وَأَسْتَجَابَ لِدُعَائِكَ !!

This issue of **MRCPPART1 MADE EASY 2017** contains the following:

- Full topic note.
- Around 2600 MCQs from **PM 2017 BANK** covering 14 chapter in internal medicine
- External links for the related guidelines (such as NICE, SIGN, RCP ...etc).
- Comments with illustrative pictures and links to external media.
- Most Recommended QBank for MRCP Part 1, Arab Board, Jordanian Board and other internal medicine assessment exams.

Please Note that

Each chapter begins with notes section. These notes are repeated under the Questions in the website. In these collections the notes are not repeated under each question except in some to avoid unnecessary repetition. For clarity and convenience the title of the related note is only mentioned after the comments in case the reader want to refer back to the respective note in the notes section.

The illustration below is to show and Example

☐	Minimal change disease
☐	Post-streptococcal glomerulonephritis
☐	Goodpasture's syndrome

Goodpasture's syndrome

- IgG deposits on renal biopsy
- anti-GBM antibodies

• These changes are characteristic of Goodpasture's syndrome.

[Discuss and give feedback](#)

Goodpasture's syndrome 

Comment of the Q

title of the note in case the reader want to refer back to the notes sections

MRCPPART1 MADE EASY 2017

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إعداد وتنسيق

M. HABAYEB

**Msc Internal. Medicine
Cairo University**

&

A. MURAD

لا تنسونا من صالح دعائكم

ما كان من خطأ فمن أنفسنا وما كان من توفيق فمن الله

تم بحمد الله وتوفيقه ومنه

جعل الله عملنا متقبلاً خالصاً لوجهه الكريم

Reference ranges

Reference ranges vary according to individual labs. All values are for adults unless otherwise stated

Full blood count

Haemoglobin	Men: 13.5-18 g/dl Women: 11.5-16 g/dl
Mean cell volume	82-100 fl
Platelets	150-400 * 10 ⁹ /l
White blood cells	4-11 * 10 ⁹ /l

Urea and electrolytes

Sodium	135-145 mmol/l
Potassium	3.5 - 5.0 mmol/l
Urea	2.0-7 mmol/l
Creatinine	55-120 umol/l
Bicarbonate	22-28 mmol/l
Chloride	95-105 mmol/l

Liver function tests

Bilirubin	3-17 umol/l
Alanine transferase (ALT)	3-40 iu/l
Aspartate transaminase (AST)	3-30 iu/l
Alkaline phosphatase (ALP)	30-100 umol/l
Gamma glutamyl transferase (yGT)	8-60 u/l
Albumin	35-50 g/l
Total protein	60-80 g/l

Other haematology

Erythrocyte sedimentation rate (ESR)	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Prothrombin time (PT)	10-14 secs
Activated partial thromboplastin time (APTT)	25-35 secs
Ferritin	20-230 ng/ml
Vitamin B12	200-900 ng/l
Folate	3.0 nmol/l
Reticulocytes	0.5-1.5%
D-Dimer	< 400 ng/ml

Other biochemistry

Calcium	2.1-2.6 mmol/l
Phosphate	0.8-1.4 mmol/l
CRP	< 10 mg/l
Thyroid stimulating hormone (TSH)	0.5-5.5 mu/l
Free thyroxine (T4)	9-18 pmol/l
Total thyroxine (T4)	70-140 nmol/l
Amylase	70-300 u/l

Uric acid	0.18-0.48 mmol/l
-----------	------------------

Arterial blood gases

pH	7.35 - 7.45
pCO2	4.5 - 6.0 kPa
pO2	10 - 14 kPa

Lipids

Desirable lipid values depend on other risk factors for cardiovascular disease, below is just a guide:

Total cholesterol	< 5 mmol/l
Triglycerides	< 2 mmol/l
HDL cholesterol	> 1 mmol/l
LDL cholesterol	< 3 mmol/l

Chapter: Respiratory medicine

Allergic bronchopulmonary aspergillosis	Notes
Alpha-1 antitrypsin deficiency	Notes
Antibiotic guidelines	Notes
ARDS	Notes
Asbestos and the lung	Notes
Aspergilloma	Notes
Asthma: acute severe	Notes
Asthma: diagnosis	Notes
Asthma: management in adults	Notes
Asthma: occupational	Notes
Bilateral hilar lymphadenopathy	Notes
Bronchiectasis: causes	Notes
Bronchiectasis: management	Notes
Chest x-ray: cavitating lung lesion	Notes
Churg-Strauss syndrome	Notes
COPD: investigation and diagnosis	Notes
COPD: long-term oxygen therapy	Notes
COPD: management of acute exacerbations	Notes
COPD: stable management	Notes
Cryptogenic organizing pneumonia	Notes
Cystic fibrosis: diagnosis	Notes
Cystic fibrosis: features	Notes
Cystic fibrosis: management	Notes
Extrinsic allergic alveolitis	Notes
Fitness to fly	Notes
Flow volume loop	Notes
Idiopathic pulmonary fibrosis	Notes

Invasive Aspergillosis	Notes
Kartagener's syndrome	Notes
<i>Legionella</i>	Notes
Lofgren's syndrome	Notes
Lung cancer: carcinoid	Notes
Lung cancer: non-small cell	Notes
Lung cancer: non-small cell management	Notes
Lung cancer: paraneoplastic features	Notes
Lung cancer: risk factors	Notes
Lung cancer: small cell	Notes
Lung fibrosis	Notes
Mesothelioma	Notes
<i>Mycoplasma pneumoniae</i>	Notes
Non-invasive ventilation	Notes
Obstructive sleep apnoea/hypopnoea syndrome	Notes
Oxygen therapy	Notes
Pleural effusion	Notes
Pleural effusion: investigation	Notes
Pneumonia: community-acquired	Notes
Pneumonia: prognostic factors	Notes
Pneumothorax	Notes
Pregnancy: DVT/PE investigation	Notes
Pulmonary arterial hypertension: causes and classification	Notes
Pulmonary embolism: investigation	Notes
Pulmonary embolism: management	Notes
Pulmonary eosinophilia	Notes
Pulmonary function tests	Notes

Chapter: Respiratory medicine

Respiratory acidosis	Notes
Respiratory alkalosis	Notes
Respiratory pathogens	Notes
Rheumatoid arthritis: respiratory manifestations	Notes
Sarcoidosis: investigation	Notes
Sarcoidosis: management	Notes
Silicosis	Notes
Transfer factor	Notes
Tuberculosis: drug therapy	Notes
Tuberculosis: screening	

Allergic bronchopulmonary aspergillosis

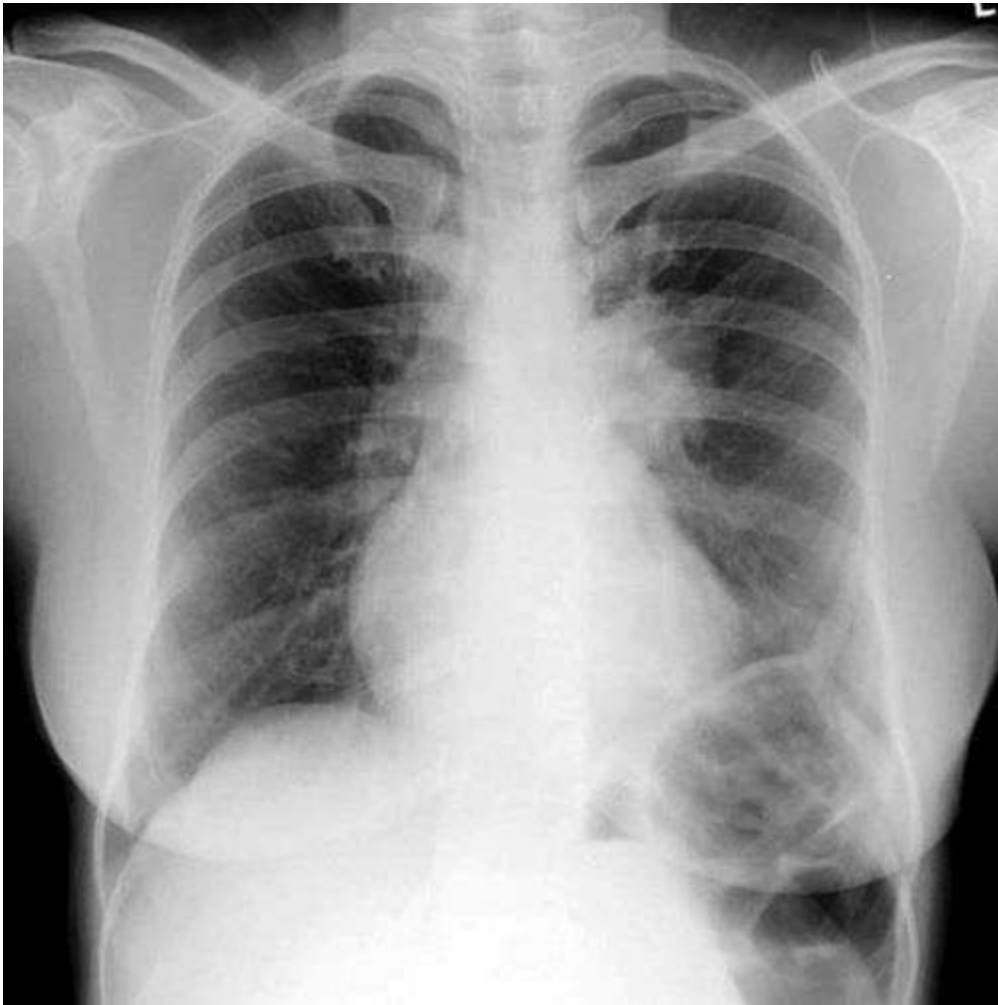
Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus* spores. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

- bronchoconstriction: wheeze, cough, dyspnoea
- bronchiectasis (proximal)

Investigations

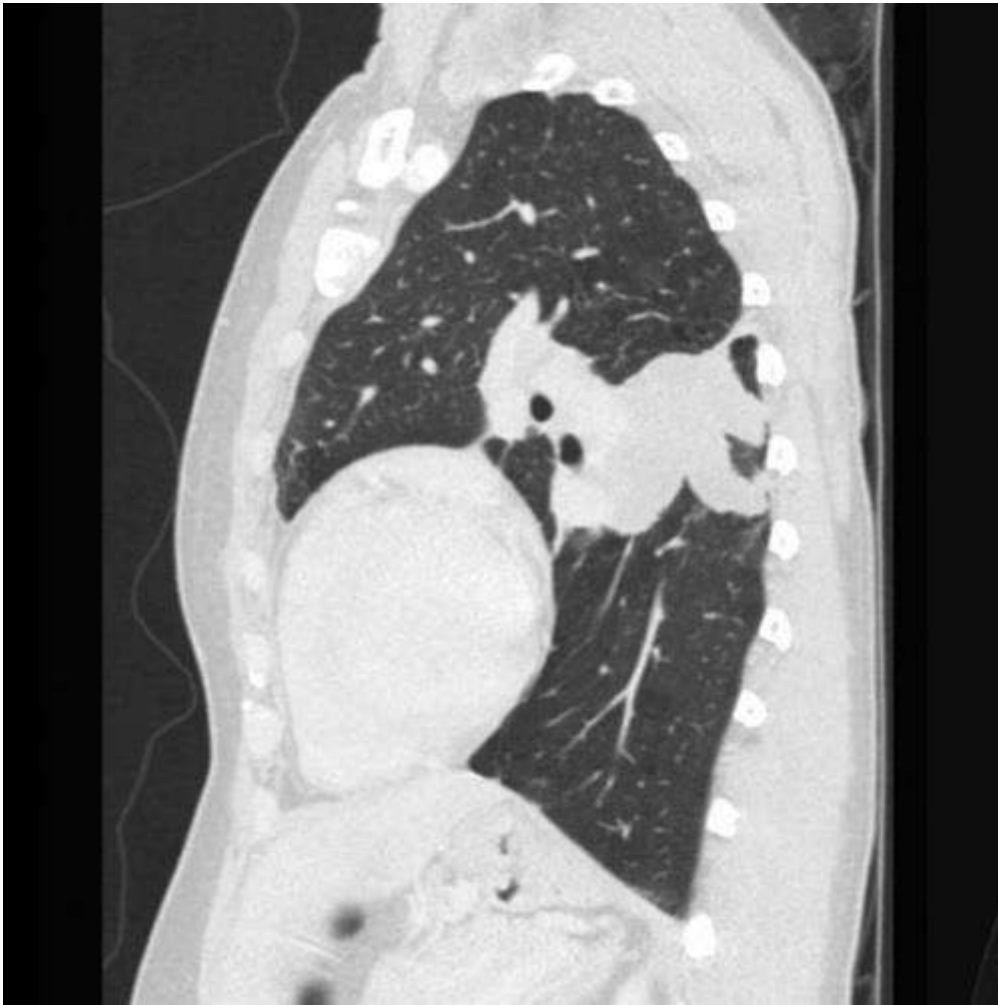
- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to *Aspergillus*
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE



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Chest x-ray of a 40-year-old woman with ABPA demonstrating a mass overlying the left hilum. In the right upper parahilar region a few ring shadow / tram track opacities are also noted, suggestive of bronchiectasis



© Image used on license from [Radiopaedia](#)



CT scan from the same patient. CT reveals a branching lesion in the superior segment of the left lower lobe with classic finger in glove appearance which represents of mucous filling dilated bronchi (i.e. bronchocoeles). Bronchocoeles are a common feature of allergic bronchopulmonary aspergillosis (ABPA)

Management

- steroids
- itraconazole is sometimes introduced as a second line agent

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal = PiMM
- homozygous PiSS (50% normal A1AT levels)
- homozygous PiZZ (10% normal A1AT levels)

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

Antibiotic guidelines

The following is based on current BNF guidelines:

Respiratory system

Condition	Recommended treatment
Exacerbations of chronic bronchitis	Amoxicillin or tetracycline or clarithromycin
Uncomplicated community-acquired pneumonia	Amoxicillin (Doxycycline or clarithromycin in penicillin allergic, add flucloxacillin if staphylococci suspected e.g. In influenza)
Pneumonia possibly caused by atypical pathogens	Clarithromycin
Hospital-acquired pneumonia	Within 5 days of admission: co-amoxiclav or cefuroxime More than 5 days after admission: piperacillin with tazobactam OR a broad-spectrum cephalosporin (e.g. ceftazidime) OR a quinolone (e.g. ciprofloxacin)

Urinary tract

Condition	Recommended treatment
Lower urinary tract infection	Trimethoprim or nitrofurantoin. Alternative: amoxicillin or cephalosporin
Acute pyelonephritis	Broad-spectrum cephalosporin or quinolone
Acute prostatitis	Quinolone or trimethoprim

Skin

Condition	Recommended treatment
Impetigo	Topical fusidic acid, oral flucloxacillin or erythromycin if widespread
Cellulitis	Flucloxacillin (clarithromycin or clindomycin if penicillin-allergic)
Erysipelas	Phenoxymethylpenicillin (erythromycin if penicillin-allergic)
Animal or human bite	Co-amoxiclav (doxycycline + metronidazole if penicillin-allergic)
Mastitis during breast-feeding	Flucloxacillin

Ear, nose & throat

Condition	Recommended treatment
Throat infections	Phenoxymethylpenicillin (erythromycin alone if penicillin-allergic)
Sinusitis	Amoxicillin or doxycycline or erythromycin
Otitis media	Amoxicillin (erythromycin if penicillin-allergic)
Otitis externa*	Flucloxacillin (erythromycin if penicillin-allergic)
Periapical or periodontal abscess	Amoxicillin
Gingivitis: acute necrotising ulcerative	Metronidazole

Genital system

Condition	Recommended treatment
Gonorrhoea	Intramuscular ceftriaxone + oral azithromycin
<i>Chlamydia</i>	Doxycycline or azithromycin
Pelvic inflammatory disease	Oral ofloxacin + oral metronidazole or intramuscular ceftriaxone + oral doxycycline + oral metronidazole
Syphilis	Benzathine benzylpenicillin or doxycycline or erythromycin
Bacterial vaginosis	Oral or topical metronidazole or topical clindamycin

Gastrointestinal

Condition	Recommended treatment
<i>Clostridium difficile</i>	First episode: metronidazole Second or subsequent episode of infection: vancomycin
Campylobacter enteritis	Clarithromycin
Salmonella (non-typhoid)	Ciprofloxacin
Shigellosis	Ciprofloxacin

*a combined topical antibiotic and corticosteroid is generally used for mild/moderate cases of otitis externa

External Links

[BNF](#)

5.1 Summary of antibacterial therapy

ARDS

Basics

- acute respiratory distress syndrome
- caused by increased permeability of alveolar capillaries leading to fluid accumulation in alveoli i.e. non-cardiogenic pulmonary oedema

Criteria (American-European Consensus Conference):

- acute onset
- bilateral infiltrates on CXR
- non-cardiogenic (pulmonary artery wedge pressure needed if doubt)
- $pO_2/FiO_2 < 200$ mmHg

Causes:

- infection: sepsis, pneumonia
- massive blood transfusion
- trauma
- smoke inhalation
- pancreatitis
- cardio-pulmonary bypass

External Links

[Royal College of Physicians](#)

2011 Acute lung injury review

Asbestos and the lung

Asbestos can cause a variety of lung disease from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They are the most common form of asbestos related lung disease and generally occur after a latent period of 20-40 years.

Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis. As with other forms of lung fibrosis the most common symptoms are shortness-of-breath and reduced exercise tolerance.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features:

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Asbestos exposure is a risk factor for lung cancer and also has a synergistic effect with cigarette smoke.

Aspergilloma

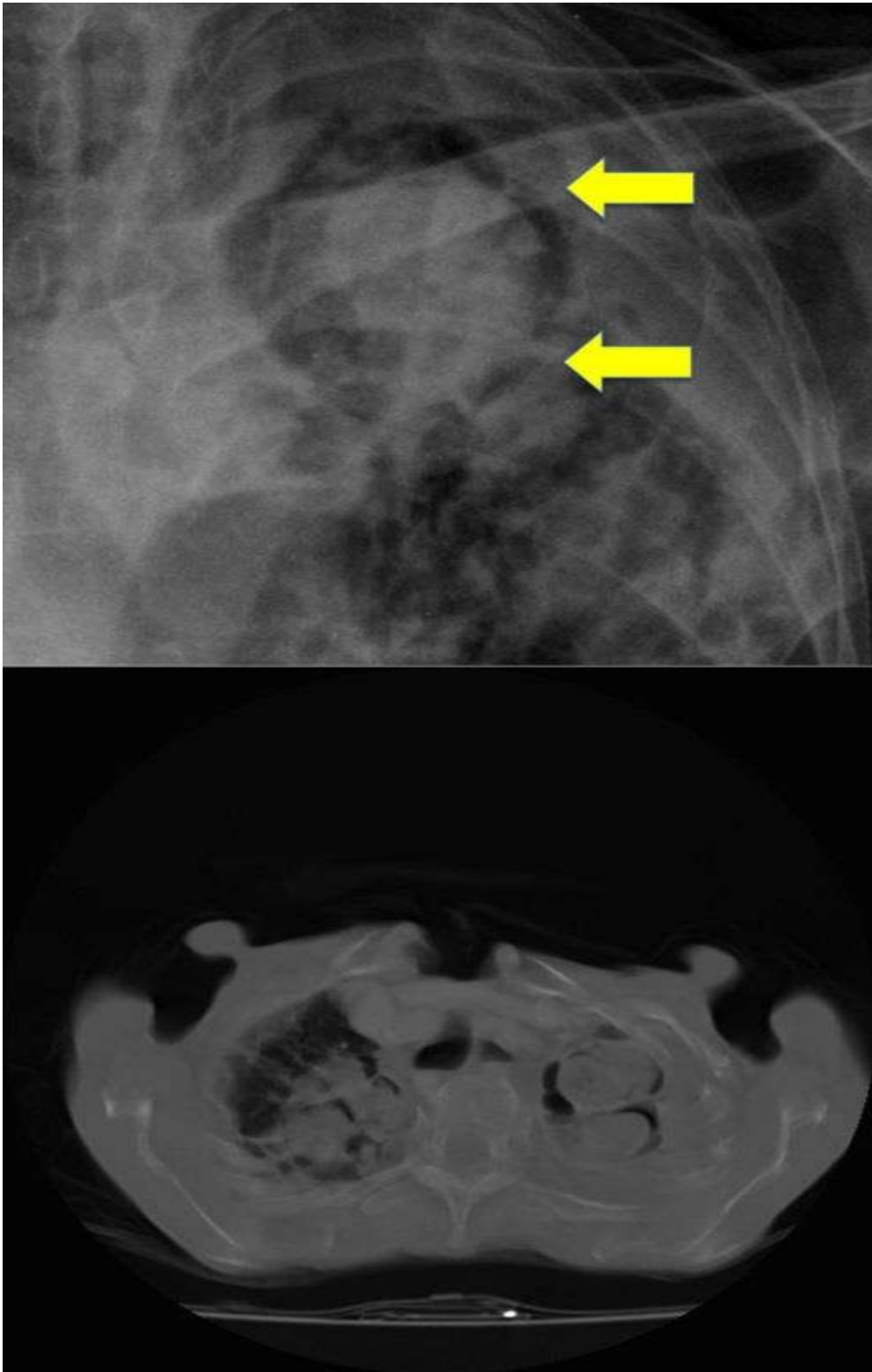
An aspergilloma is a mycetoma (mass-like fungus ball) which often colonises an existing lung cavity (e.g. secondary to tuberculosis, lung cancer or cystic fibrosis)

Usually asymptomatic but features may include:

- cough
- haemoptysis (may be severe)

Investigations

- chest x-ray containing a rounded opacity
- high titres Aspergillus precipitins



© Image used on license from [Radiopaedia](#)



Aspergilloma in a patient with cavities secondary to previous tuberculosis infection. The close-up CXR and CT scan from the same patient demonstrate a rounded soft tissue attenuating masses located in a surrounding cavity.

Asthma: acute severe

Patients with acute severe asthma are stratified into moderate, severe or life-threatening

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

British Thoracic Society guidelines

- magnesium sulphate recommended as next step for patients who are not responding (e.g. 1.2 - 2g IV over 20 mins)
- little evidence to support use of IV aminophylline (although still mentioned in management plans)
- if no response consider IV salbutamol

External Links

[British Thoracic Society](#)

2016 Asthma guidelines

Asthma: diagnosis

The British Thoracic Society (BTS) updated their asthma guidelines in 2016 resulting in some significant changes in how asthma is diagnosed and managed.

The diagnosis of asthma remains clinical, based on a combination of history, examination and investigation results. **There is no one definitive test and the combination of findings results in a high, intermediate or low probability of asthma.**

There are a number of respiratory symptoms which should make you consider asthma as a diagnosis. The guidelines remind us that the predictive value of individual symptoms or signs is poor.

- wheeze
- cough
- breathlessness
- chest tightness

Once a diagnosis is being considered, a combination of history, examination and investigation results should be used to determine whether a patient has a high, intermediate or low probability of asthma. **Factors which should be considered include:**

- **recurrent episodes of symptoms:** may be triggered by viral infection, allergen exposure, NSAIDs/beta-blockers and/or exacerbated by exercise, cold air and emotion/laughter in children
- **recorded observation of wheeze:** due to varying use of language this usually means wheeze documented by a clinician
- **symptom variability:** asthma is generally worse at night or early in the morning
- **personal history of atopy:** e.g. eczema/allergic rhinitis
- **absence of symptoms of alternative diagnosis:** e.g. COPD, dysfunctional breathing or obesity*
- **historical record of variable peak flows or FEV₁**

At this point, **the guidelines do not make specific recommendations about who is considered to have a high, medium or low probability of asthma.** It is now recommended that we make a clinical judgement about likelihood, which probably better reflects day-to-day practice.

If a patient has a **high** probability of asthma:

- treatment should be initiated (typically 6 weeks of inhaled corticosteroid)
- an assessment should be made about whether the patient has responded to treatment using either a validated scoring system or lung functions tests (FEV₁ at clinic visits or by domiciliary serial peak flows)
- a good response to treatment is clearly supportive of an asthma diagnosis, a poor response should prompt tests for airway obstruction with spirometry and bronchodilator reversibility

If a patient has a **intermediate** probability of asthma:

- tests for airway obstruction with spirometry and bronchodilator reversibility should be undertaken
- if a child is too young to perform spirometry the BTS recommend we consider watchful waiting if the child is asymptomatic, or offer a carefully monitored trial of treatment if the child is symptomatic

If a patient has a **low** probability of asthma:

- investigations should continue looking for alternative causes of the presenting symptoms
- if other diagnoses considered unlikely then investigations for asthma should be arranged, including spirometry and bronchodilator reversibility

Reasons for referring to secondary care include:

- if the diagnosis remains unclear despite investigations
- suspected occupational asthma
- poor response to treatment
- a severe or life-threatening asthma attack
- there are other 'red-flags' which may indicate more severe disease or alternative diagnosis such as systemic features (weight loss, fever etc), failure to thrive in children, focal signs, chronic sputum production etc. Please see the guidelines for the full list

Once a diagnosis of asthma has been made all patients (or parents/carers) should be offered self-management education including a written personalised asthma action plan.

- in adults, these may be based on symptoms/peak flows whereas in children symptom-based plans are preferred

*in the extended guidelines the BTS state some of the symptoms/factors which may point to an alternative diagnosis. **Please see the guidelines for the full list:**

Clinical clue	Possible diagnosis
Predominant cough without lung function abnormalities	Chronic cough syndromes; pertussis
Prominent dizziness, light-headedness, peripheral tingling	Dysfunctional breathing
Recurrent severe asthma attacks without objective confirmatory evidence	Vocal cord dysfunction
Predominant nasal symptoms without lung function abnormalities	Rhinitis
Postural and food-related symptoms, predominant cough	Gastro-oesophageal reflux
Orthopnoea, paroxysmal nocturnal dyspnoea, peripheral oedema, preexisting cardiac disease	Cardiac failure
Crackles on auscultation	Pulmonary fibrosis
Significant smoking history (ie, >30 pack-years), age of onset >35 years	COPD
Chronic productive cough in the absence of wheeze or breathlessness	Bronchiectasis; inhaled foreign body; obliterative bronchioitis; large airway stenosis
New onset in smoker, systemic symptoms, weight loss, haemoptysis	Lung cancer; sarcoidosis

External Links

[British Thoracic Society](#)

2016 Asthma guidelines

Asthma: management in adults

The British Thoracic Society (BTS) updated their asthma guidelines in 2016 resulting in some significant changes in how asthma is diagnosed and managed. **In terms of management, some of the key changes include:**

- the previous numbered steps have been abandoned
- all patients should receive an inhaled corticosteroid, right from the point of assessment in patients with suspected asthma
- a combined inhaled corticosteroid/long-acting beta agonist (ICS/LABA) is recommended instead of separate inhalers to increase compliance
- a long-acting muscarinic antagonist (LAMA) is an options for patient who haven't responded to a combined ICS/LABA inhaler

In terms of management, the BTS suggest the following approach:

- 1. Start treatment at the level most appropriate to initial severity
- 2. Achieve early control
- 3. Maintain control by increasing treatment as necessary and decreasing treatment when control is good

The previous steps 1-5 of asthma management have been abandoned. Previously 'Step 1' of asthma management was the use of a short-acting beta agonist (SABA) as required. This is no longer suggested as the initial first step. Instead, **all patients from the 'diagnosis and assessment' onwards should use a SABA as required.** Having to use a SABA more than **3 times per week** is considered a sign that further add-on therapy is needed.

Initial step

It is now recommended that a low-dose inhaled corticosteroid (ICS) is started in all patients with a diagnosis, or suspected diagnosis, of asthma, right from the 'diagnosis and assessment' period prior to a formal diagnosis being made. **The first step of asthma management is now a low-dose inhaled corticosteroid in combination with a short-acting beta agonist.**

Next step.

The **a long-acting beta agonist (LABA).** This should ideally be in the form of **a combination inhaler.**

Next step..

If poor control remains the next step depends on the response to the LABA:

- no response to LABA - stop LABA and increase dose of ICS to medium-dose
- response to LABA - continue LABA and increase ICS to medium-dose. An alternative to this is to continue on the current treatment but consider a trial of a leukotriene receptor antagonist, SR theophylline or a long-acting muscarinic antagonist (LAMA)

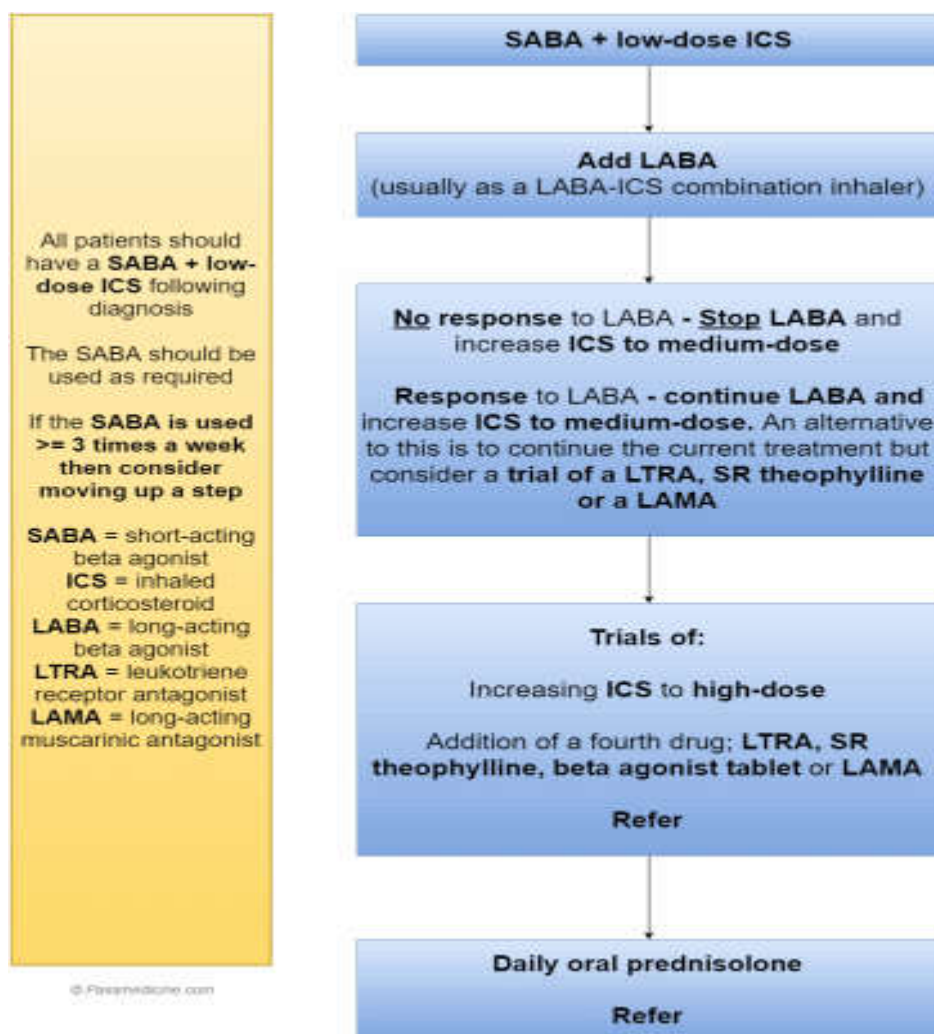
Next step...

Consider trials of either:

- increasing the ICS to high-dose, OR
- the addition of a fourth drug (e.g. a leukotriene receptor antagonist, SR theophylline, a LAMA or an oral beta-agonist tablet)
- patient should be referred to specialist care at this point

Next step....

The consideration of regular oral steroids at the lowest dose providing adequate daily control.



2016 British Thoracic Society asthma algorithm for adults

Table showing examples of inhaled corticosteroid doses

Low	Medium	High
Beclometasone dipropionate - Clenil - 100 mcg two puffs bd - Qvar - 50 mcg two puffs bd	Beclometasone dipropionate - Clenil - 200 mcg two puffs bd - Qvar - 100 mcg two puffs bd	Beclometasone dipropionate - Clenil - 250 mcg two-four puffs bd - Qvar - 100 mcg four puffs bd
Fluticasone propionate Flixotide - 50 mcg two puffs bd	Fluticasone propionate Flixotide - 125 mcg two puffs bd	Fluticasone propionate Flixotide - 250 mcg two puffs bd

[British Thoracic Society](#)

2016 Asthma guidelines

Asthma: occupational

Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.

Exposure to the following chemicals is associated with occupational asthma:

- isocyanates - the most common cause. Example occupations include spray painting and foam moulding using adhesives
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

Serial measurements of peak expiratory flow are recommended at work and away from work.

Referral should be made to a respiratory specialist for patients with suspected occupational asthma.

External Links

[British Thoracic Society](#)

2014 Asthma guidelines

[Royal College of Physicians](#)

2012 Concise guidance: diagnosis, management and prevention of occupational asthma

Bilateral hilar lymphadenopathy

The most common causes of bilateral hilar lymphadenopathy are sarcoidosis and tuberculosis

Other causes include:

- lymphoma/other malignancy
 - pneumoconiosis e.g. berylliosis
 - fungi e.g. histoplasmosis, coccidioidomycosis
-

Bronchiectasis: causes

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. There are a wide variety of causes are listed below:

Causes:

- post-infective: tuberculosis, measles, pertussis, pneumonia
- cystic fibrosis
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: selective IgA, hypogammaglobulinaemia
- allergic bronchopulmonary aspergillosis (ABPA)
- ciliary dysketic syndromes: Kartagener's syndrome, Young's syndrome
- yellow nail syndrome



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Chest x-ray showing tramlines, most prominent in the left lower zone



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CT chest showing widespread tram-track and signet ring signs

External Links

[British Thoracic Society](#)

2010 Guideline for non-CF bronchiectasis

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. **After assessing for treatable causes (e.g. immune deficiency) management is as follows:**

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease).

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- *Klebsiella* spp.
- *Streptococcus pneumoniae*

External Links

[British Thoracic Society](#)

2010 Bronchiectasis guideline

[Clinical Knowledge Summaries](#)

Bronchiectasis guidelines

Chest x-ray: cavitating lung lesion
--

Differential

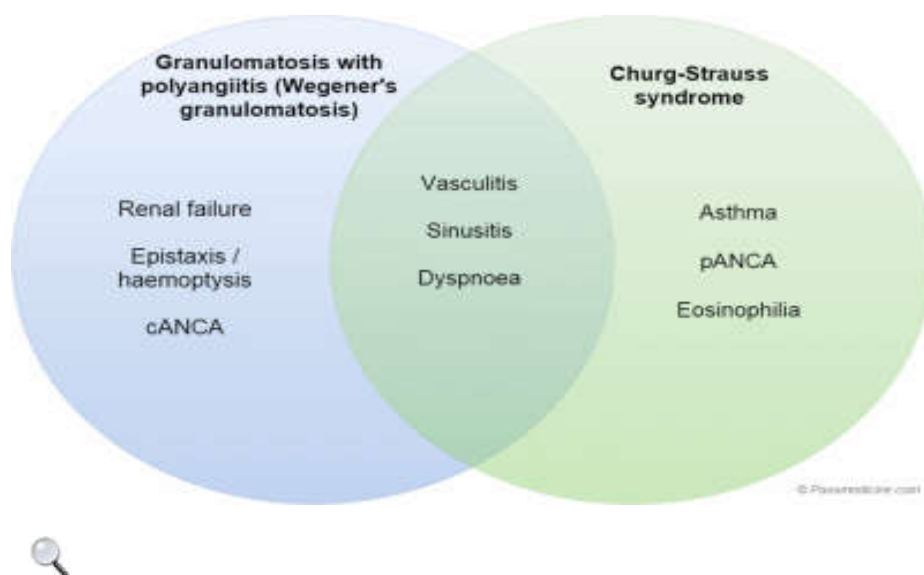
- abscess (*Staph aureus*, *Klebsiella* and *Pseudomonas*)
 - squamous cell lung cancer
 - tuberculosis
 - Wegener's granulomatosis
 - pulmonary embolism
 - rheumatoid arthritis
 - aspergillosis, histoplasmosis, coccidioidomycosis
-

Churg-Strauss syndrome

Churg-Strauss syndrome is an ANCA associated small-medium vessel vasculitis.

Features:

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease

COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild**
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

**symptoms should be present to diagnose COPD in these patients

External Links

[NICE](#)

2010 COPD guidelines

COPD: long-term oxygen therapy

The 2010 NICE guidelines on COPD clearly define which patients should be assessed for and offered long-term oxygen therapy (LTOT). Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day. Oxygen concentrators are used to provide a fixed supply for LTOT.

Assess patients if any of the following:

- very severe airflow obstruction (FEV1 < 30% predicted). Assessment should be 'considered' for patients with severe airflow obstruction (FEV1 30-49% predicted)
- cyanosis
- polycythaemia
- peripheral oedema
- raised jugular venous pressure
- oxygen saturations less than or equal to 92% on room air

Assessment is done by measuring arterial blood gases on 2 occasions at least 3 weeks apart in patients with stable COPD on optimal management.

Offer LTOT to patients with a pO₂ of < 7.3 kPa or to those with a pO₂ of 7.3 - 8 kPa and one of the following:

- secondary polycythaemia
- nocturnal hypoxaemia
- peripheral oedema
- pulmonary hypertension

External Links

[NICE](#)

2010 COPD guideline

[NICE](#)

2010 COPD guidelines

[British Thoracic Society](#)

2015 BTS Guidelines for Home Oxygen Use in Adults

COPD: management of acute exacerbations

The most common bacterial organisms that cause infective exacerbations of COPD are:

- *Haemophilus influenzae* (most common cause)
- *Streptococcus pneumoniae*
- *Moraxella catarrhalis*

Respiratory viruses account for around 30% of exacerbations, with the human rhinovirus being the most important pathogen.

NICE guidelines from 2010 recommend the following:

- increase frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 7-14 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'

External Links

[NICE](#)

2010 COPD guidelines

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2010.

General management:

- smoking cessation advice
- annual influenza vaccination
- one-off pneumococcal vaccination

Bronchodilator therapy:

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by the FEV1

FEV1 > 50%

- long-acting beta2-agonist (LABA), for example salmeterol, or:
- long-acting muscarinic antagonist (LAMA), for example tiotropium

FEV1 < 50%

- LABA + inhaled corticosteroid (ICS) in a combination inhaler, or:
- LAMA

For patients with persistent exacerbations or breathlessness

- if taking a LABA then switch to a LABA + ICS combination inhaler
- otherwise give a LAMA and a LABA + ICS combination inhaler

Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot use inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE.

Factors which may improve survival in patients with stable COPD:

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

External Links

[NICE](#)

2010 COPD guidelines

[Global Initiative for Chronic Obstructive Lung Disease](#)

GOLD criteria 2017

Cryptogenic organizing pneumonia

Cryptogenic organizing pneumonia (COP) is a diffuse interstitial lung disease that affects the distal bronchioles, respiratory bronchioles, alveolar ducts and alveolar walls. It affects 6-7 people per 100,000. The aetiology is unknown.

Males and females are equally affected and tend to present in the 5th or 6th decade of life and is not associated with smoking. Patients typically present with a cough, shortness of breath, fever and malaise. Symptoms can be present for weeks or months. There is often a history of non-response to antibiotics. Haemoptysis is rare. Clinical examination is often normal but inspiratory crackles can be heard. Wheeze and clubbing are rare.

Bloods show a leukocytosis and an elevated ESR and CRP. Imaging typically shows bilateral patchy or diffuse consolidative or ground glass opacities. Lung function tests are most commonly restrictive but can be obstructive or normal. The transfer factor is reduced.

Diagnosis is clinical. Treatment is watch and wait if mild or high dose oral steroids if severe.

Cystic fibrosis: diagnosis

Sweat test

- patient's with CF have abnormally high sweat chloride
- normal value < 40 mEq/l, CF indicated by > 60 mEq/l

Causes of false positive sweat test

- malnutrition
 - adrenal insufficiency
 - glycogen storage diseases
 - nephrogenic diabetes insipidus
 - hypothyroidism, hypoparathyroidism
 - G6PD
 - ectodermal dysplasia
-

Cystic fibrosis: features

Presenting features

- neonatal period (around 20%): meconium ileus, less commonly prolonged jaundice
- recurrent chest infections (40%)
- malabsorption (30%): steatorrhoea, failure to thrive
- other features (10%): liver disease

Other features of cystic fibrosis

- short stature
 - diabetes mellitus
 - delayed puberty
 - rectal prolapse (due to bulky stools)
 - nasal polyps
 - male infertility, female subfertility
-

Cystic fibrosis: management

Management of cystic fibrosis involves a multidisciplinary approach

Key points

- regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful
- high calorie diet, including high fat intake*
- vitamin supplementation
- pancreatic enzyme supplements taken with meals
- heart and lung transplant

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

External Links

[Cystic Fibrosis Trust](#)

Nutrition for adults

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins
- farmers lung: spores of *Saccharopolyspora rectivirgula* (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*.

Presentation

- acute: occur 4-8 hrs after exposure, SOB, dry cough, fever
- chronic

Investigation

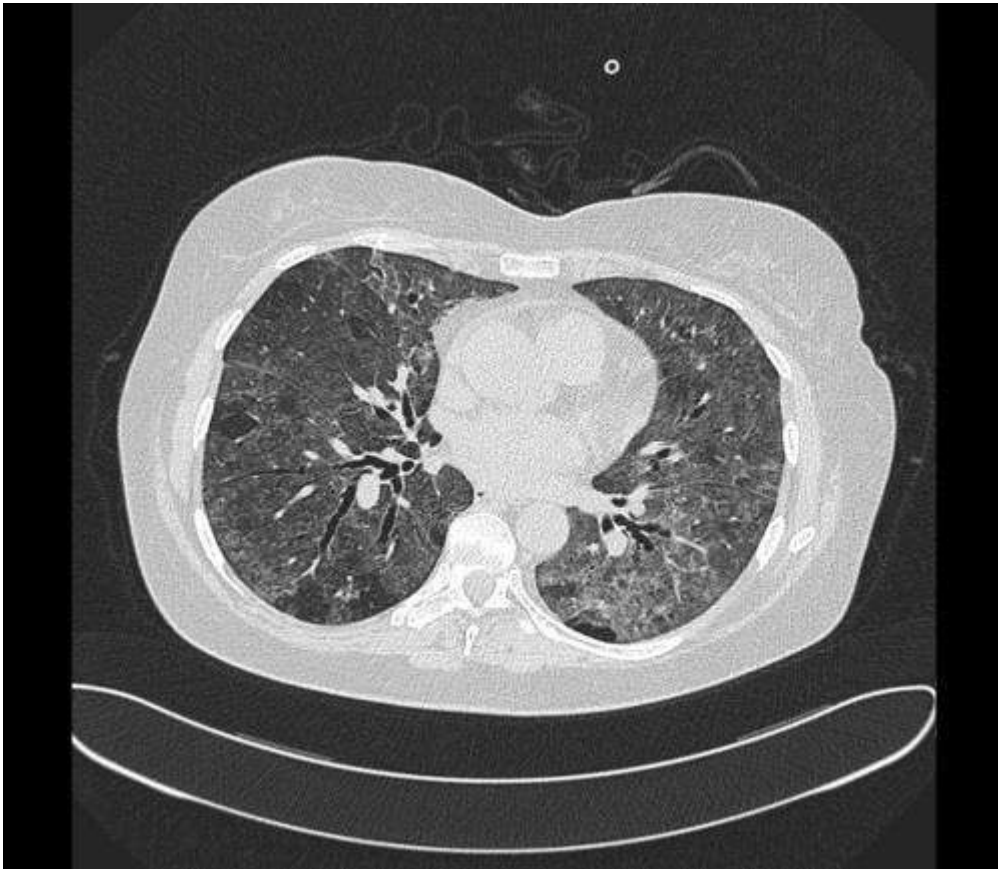
- chest x-ray: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- blood: NO eosinophilia

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*



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Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multiple centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

Fitness to fly

The Civil Aviation Authority (CAA) has issued guidelines on air travel for people with medical conditions; please see the link provided.

Cardiovascular disease

- unstable angina, uncontrolled hypertension, uncontrolled cardiac arrhythmia, decompensated heart failure, severe symptomatic valvular disease: should not fly
- uncomplicated myocardial infarction: may fly after 7-10 days
- complicated myocardial infarction: after 4-6 weeks
- coronary artery bypass graft: after 10-14 days
- percutaneous coronary intervention: after 5 days

Respiratory disease

- pneumonia: should be 'clinically improved with no residual infection'
- pneumothorax: absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Pregnancy

- most airlines do not allow travel after 36 weeks for a single pregnancy and after 32 weeks for a multiple pregnancy
- most airlines require a certificate after 28 weeks confirming that the pregnancy is progressing normally

Surgery

- travel should be avoided for 10 days following abdominal surgery
- laparoscopic surgery: after 24 hours
- colonoscopy: after 24 hours
- following the application of a plaster cast, the majority of airlines restrict flying for 24 hours on flights of less than 2 hours or 48 hours for longer flights

Haematological disorders

- patients with a haemoglobin of greater than 8 g/dl may travel without problems (assuming there is no coexisting condition such as cardiovascular or respiratory disease).

External Links

[Civil Aviation Authority](#)

Fitness to fly guidelines

Flow volume loop

A normal flow volume loop is often described as a 'triangle on top of a semi circle'

Flow volume loops are the most suitable way of assessing compression of the upper airway

External Links

[Spirometry Guru](#)

Interpretation of the Flow-Volume Loop

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF, previously termed cryptogenic fibrosing alveolitis) is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs. Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when no underlying cause exists.

IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal crackles on auscultation
- dry cough
- clubbing

Diagnosis

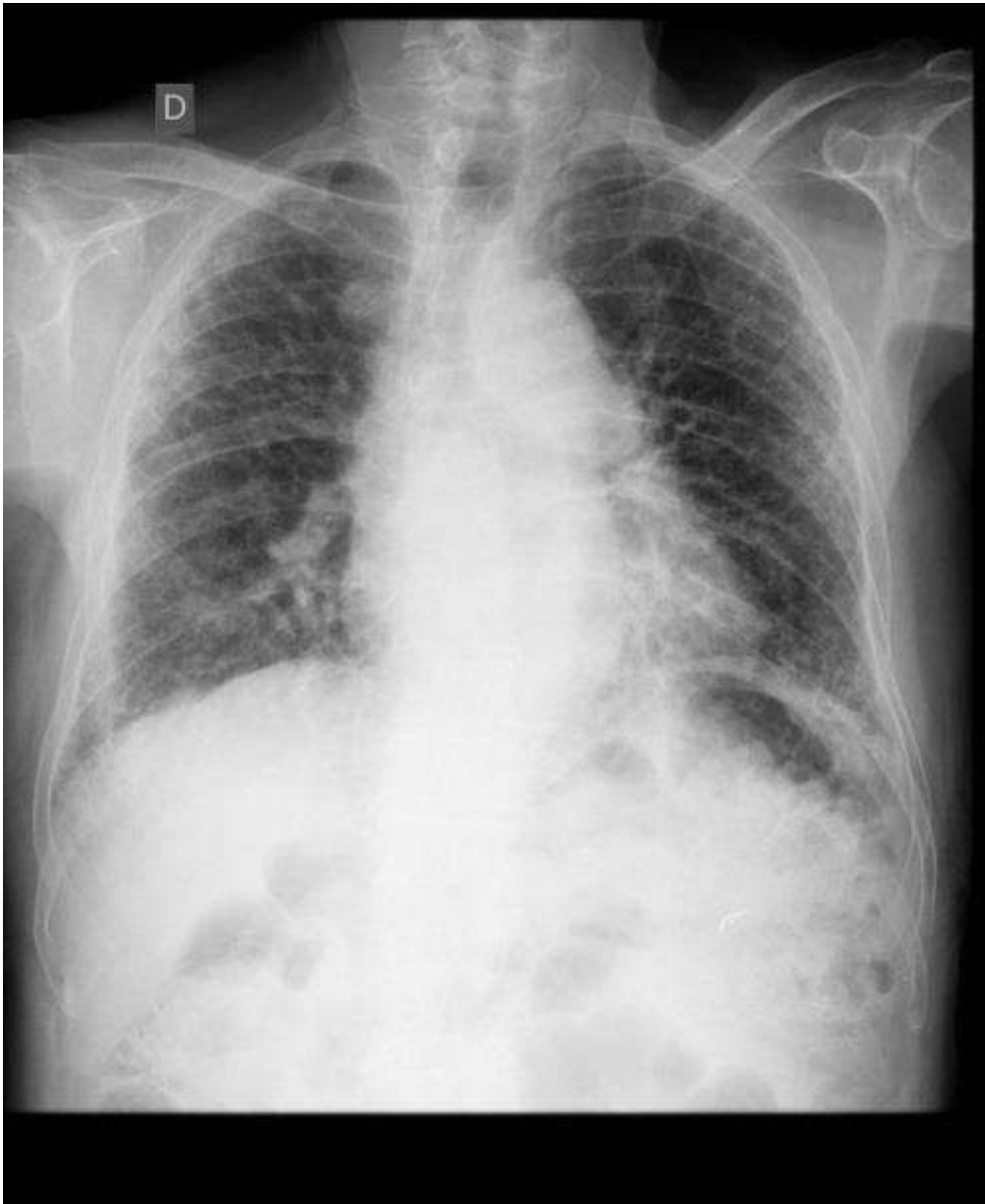
- spirometry: classically a restrictive picture (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- impaired gas exchange: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a chest x-ray but high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF
- ANA positive in 30%, rheumatoid factor positive in 10% but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. Titres are usually low

Management

- pulmonary rehabilitation
- very few medications have been shown to give any benefit in IPF. There is some evidence that pirfenidone (an antifibrotic agent) may be useful in selected patients (see NICE guidelines)
- many patients will require supplementary oxygen and eventually a lung transplant

Prognosis

- poor, average life expectancy is around 3-4 years



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Chest X-ray shows sub-pleural reticular opacities that increase from the apex to the bases of the lungs



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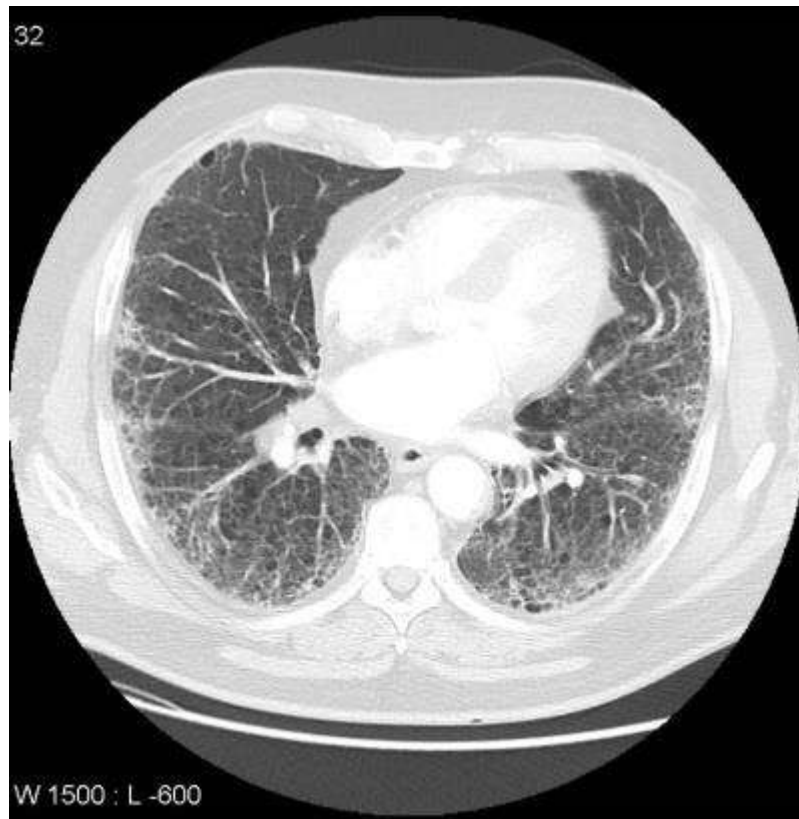




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Chest X-ray and CT scan from a patient who presented with dyspnoea. The x-ray shows reticular opacities predominantly in the bases. In addition the CT demonstrates honeycombing and traction bronchiectasis



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CT scan showing advanced pulmonary fibrosis including 'honeycombing'

External Links

[NICE](#)

2013 Idiopathic pulmonary fibrosis guidelines

Invasive Aspergillosis

Seen in the immunocompromised host to include patients with a chronic granulomatous disease, patients undergoing chemotherapy and patients receiving a bone marrow transplant.

Presentation - Pulmonary symptoms are most common, presenting with a cough, fever, haemoptysis (which can be severe), dyspnoea and pleuritic chest pain but may be atypical. There is haematogenous spread to other organs, most commonly bone resulting in osteomyelitis.

Chapter: Respiratory medicine

Investigations - can be hard to diagnose.

- ◆ Chest X-ray may show consolidation, nodules, infiltrates, or cavitating lesions.
- ◆ Chest CT may show the 'halo' sign (however aspergillosis in patients with chronic granulomatous disease typically does not produce this sign).
- ◆ Cultures can be obtained from sputum, broncho-alveolar lavage, lung tissue via trans-thoracic percutaneous biopsy. In addition, there is an assay to detect Galactomannan which a component aspergillosis cell wall.

Treatment - is with antifungals. The first line is voriconazole

ref: Centres for disease control website -

<https://www.cdc.gov/fungal/diseases/aspergillosis/treatment.html>

Kartagener's syndrome

Kartagener's syndrome (also known as primary ciliary dyskinesia) was first described in 1933 and most frequently occurs in examinations due to its association with dextrocardia (e.g. 'quiet heart sounds', 'small volume complexes in lateral leads')

Pathogenesis

- dynein arm defect results in immotile cilia

Features

- dextrocardia or complete situs inversus
 - bronchiectasis
 - recurrent sinusitis
 - subfertility (secondary to diminished sperm motility and defective ciliary action in the fallopian tubes)
-

Legionella

Legionnaire's disease is caused by the intracellular bacterium *Legionella pneumophila*. It is typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays. Person-to-person transmission is not seen

Features

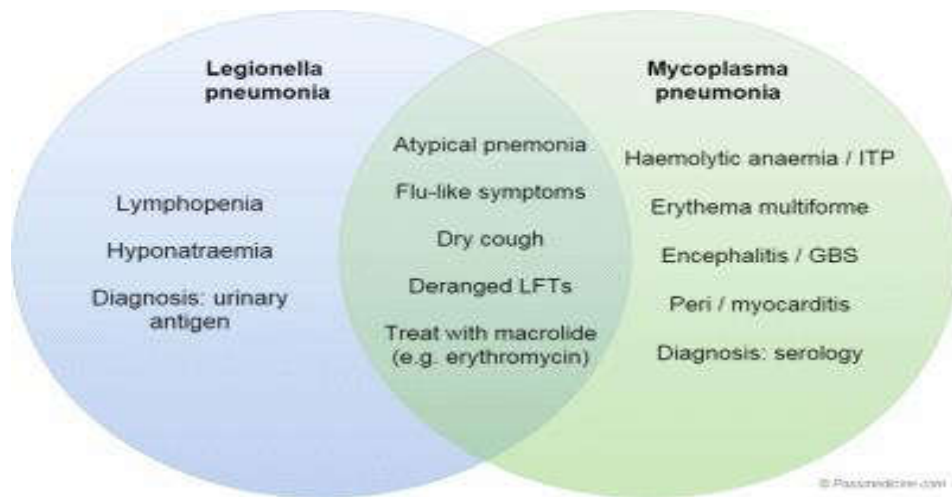
- flu-like symptoms including fever (present in > 95% of patients)
- dry cough
- relative bradycardia
- confusion
- lymphopaenia
- hyponatraemia
- deranged liver function tests
- pleural effusion: seen in around 30% of patients

Diagnosis

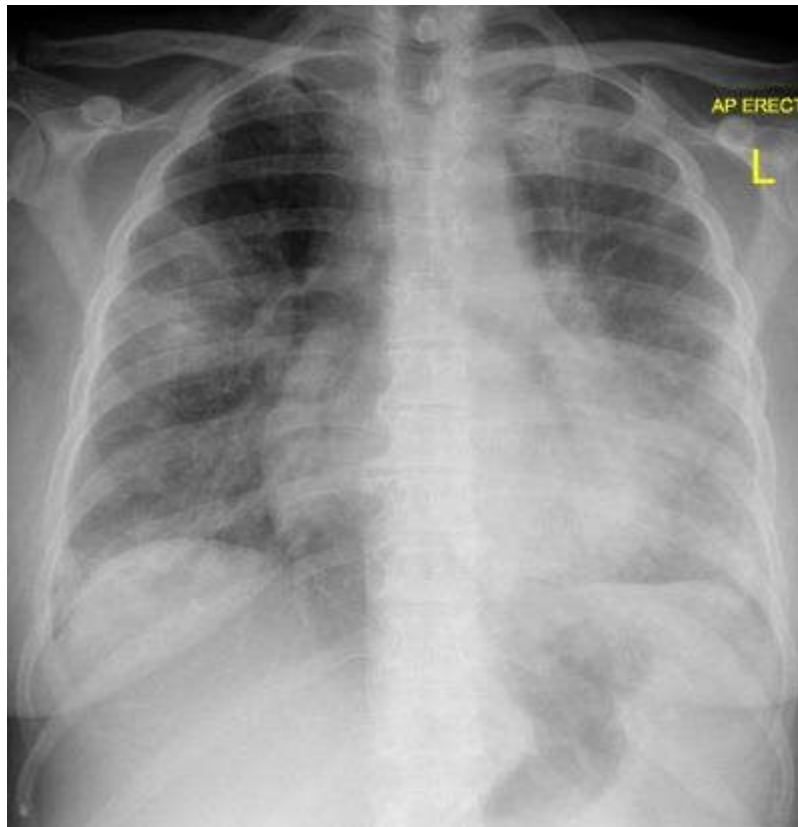
- urinary antigen

Management

- treat with erythromycin



Comparison of Legionella and Mycoplasma pneumonia



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Chest x-ray features of legionella pneumonia are non-specific but includes a mid-to-lower zone predominance of patchy consolidation. Pleural effusions are seen in around 30%.

Lofgren's syndrome

Lofgren's syndrome is an acute form sarcoidosis characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia.

It typically occurs in young females and carries an excellent prognosis.

Lung cancer: carcinoid

The vast majority of bronchial adenomas are carcinoid tumours, arising from the amine precursor uptake and decarboxylation (APUD) system, like small cell tumours. Lung carcinoid accounts 1% of lung tumours and for 10% of carcinoid tumours. The term bronchial adenoma is being phased out.

Lung carcinoid

- typical age = 40-50 years
- smoking not risk factor
- slow growing: e.g. long history of cough, recurrent haemoptysis
- often centrally located and not seen on CXR
- 'cherry red ball' often seen on bronchoscopy
- carcinoid syndrome itself is rare (associated with liver metastases)

Management

- surgical resection
 - if no metastases then 90% survival at 5 years
-

Lung cancer: non-small cell

There are three main subtypes of non-small cell lung cancer:

Squamous cell cancer

- typically central
- associated with parathyroid hormone-related protein (PTHrP) secretion → hypercalcaemia
- strongly associated with finger clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)

Adenocarcinoma

- typically peripheral
- most common type of lung cancer in non-smokers, although the majority of patients who develop lung adenocarcinoma are smokers

Large cell lung carcinoma

- typically peripheral
- anaplastic, poorly differentiated tumours with a poor prognosis
- may secrete β -hCG

Lung cancer: non-small cell management

Management

- only 20% suitable for surgery
- mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- curative or palliative radiotherapy
- poor response to chemotherapy.

Surgery contraindications

- assess general health
- stage IIIb or IV (i.e. metastases present)
- FEV1 < 1.5 litres is considered a general cut-off point*
- malignant pleural effusion
- tumour near hilum
- vocal cord paralysis
- SVC obstruction

* However if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results

External Links

[British Thoracic Society](#)

2010 BTS guidelines on lung cancer management

[SIGN](#)

2014 Lung cancer management guidelines

Lung cancer: paraneoplastic features

Small cell

- ADH
- ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome

Squamous cell

- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)
- hyperthyroidism due to ectopic TSH

Adenocarcinoma

- gynaecomastia



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Hypertrophic pulmonary osteoarthropathy is a proliferative periostitis involving that typically involves the long bones. It is often painful.

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a $10 * 5 = 50$ times increased risk

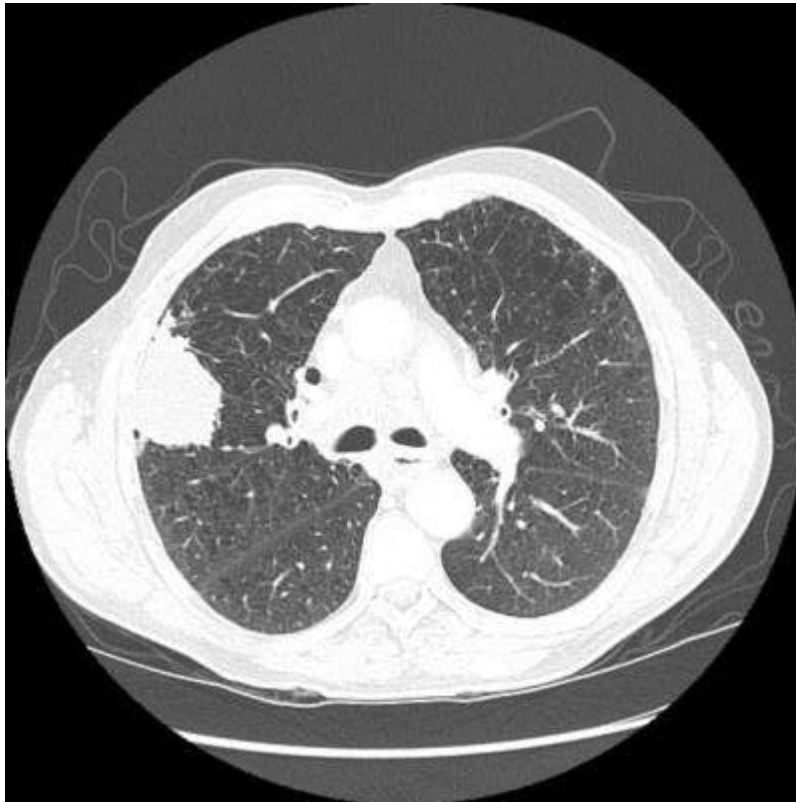
Lung cancer: small cell

Features

- usually central
- arise from APUD* cells
- associated with ectopic ADH, ACTH secretion
- ADH → hyponatraemia
- ACTH → Cushing's syndrome
- ACTH secretion can cause bilateral adrenal hyperplasia, the high levels of cortisol can lead to hypokalaemic alkalosis
- Lambert-Eaton syndrome: antibodies to voltage gated calcium channels causing myasthenic like syndrome

Management

- usually metastatic disease by time of diagnosis
- patients with very early stage disease (T1-2a, N0, M0) are now considered for surgery. NICE support this approach in their 2011 guidelines
- however, most patients with limited disease receive a combination of chemotherapy and radiotherapy
- patients with more extensive disease are offered palliative chemotherapy



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CT scan showing small cell lung cancer with multiple pulmonary nodules and extensive mediastinal nodal metastases.

**an acronym for*

- Amine - high amine content
- Precursor Uptake - high uptake of amine precursors
- Decarboxylase - high content of the enzyme decarboxylase

External Links

[NICE](#)

2011 Lung cancer guidelines

[SIGN](#)

2014 Lung cancer management guidelines

Lung fibrosis

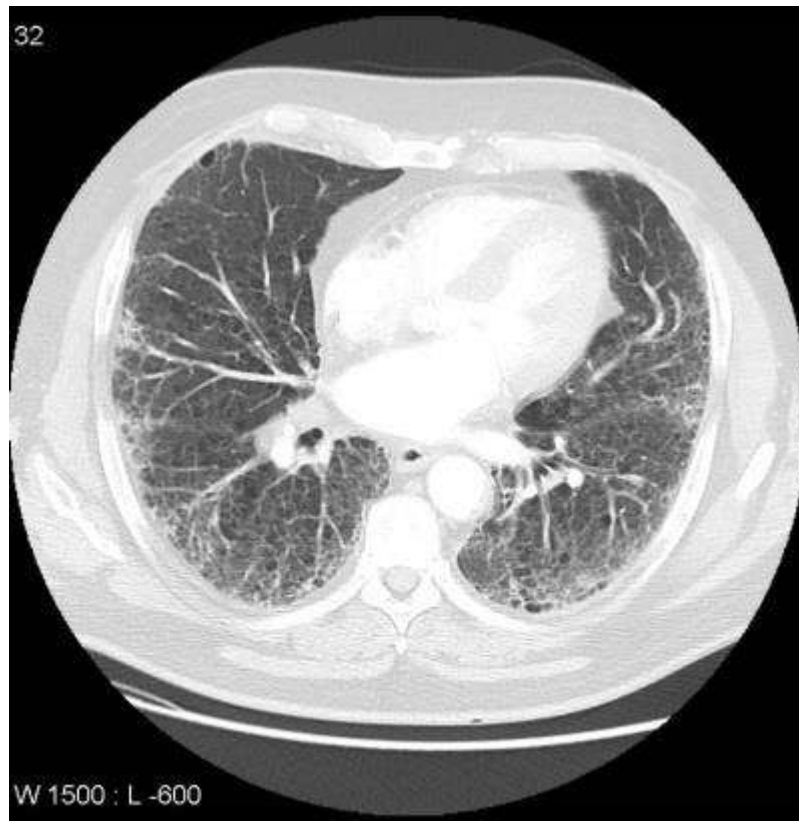
It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the **upper** zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis

Fibrosis predominately affecting the **lower** zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis)
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis



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CT scan showing advanced pulmonary fibrosis including 'honeycombing'

Mesothelioma

Features

- Dyspnoea, weight loss, chest wall pain
- Clubbing
- 30% present as painless pleural effusion
- Only 20% have pre-existing asbestosis
- History of asbestos exposure in 85-90%, latent period of 30-40 years

Basics

- Malignancy of mesothelial cells of pleura
- Metastases to contralateral lung and peritoneum
- Right lung affected more often than left

Investigation/diagnosis

- suspicion is normally raised by a chest x-ray showing either a pleural effusion or pleural thickening
- the next step is normally a pleural CT
- if a pleural effusion is present fluid should be sent for MC&S, biochemistry and cytology (but cytology is only helpful in 20-30% of cases)
- local anaesthetic thoracoscopy is increasingly used to investigate cytology negative exudative effusions as it has a high diagnostic yield (around 95%)
- if an area of pleural nodularity is seen on CT then an image-guided pleural biopsy may be used

Management

- Symptomatic
- Industrial compensation
- Chemotherapy, Surgery if operable
- Prognosis poor, median survival 12 months

External Links

[British Thoracic Society](#)

2010 Pleural disease guidelines

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune haemolytic anaemia. Epidemics of *Mycoplasma pneumoniae* classically occur every 4 years. It is important to recognise atypical pneumonias as they may not respond to penicillins or cephalosporins due to it lacking a peptidoglycan cell wall.

Features:

- the disease typically has a prolonged and gradual onset
- flu-like symptoms classically precede a dry cough
- bilateral consolidation on x-ray
- complications may occur as below

Complications

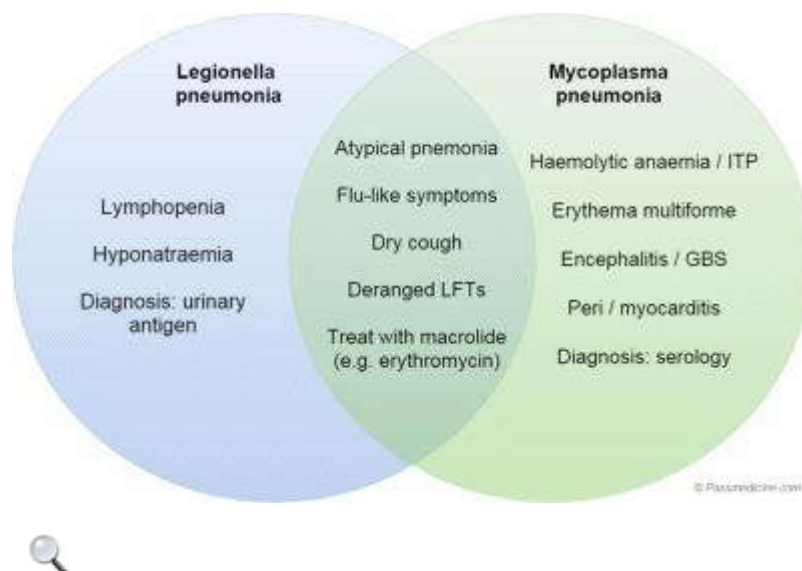
- cold agglutins (IgM) may cause an haemolytic anaemia, thrombocytopenia
- erythema multiforme, erythema nodosum
- meningoencephalitis, Guillain-Barre syndrome
- bullous myringitis: painful vesicles on the tympanic membrane
- pericarditis/myocarditis
- gastrointestinal: hepatitis, pancreatitis
- renal: acute glomerulonephritis

Investigations

- diagnosis is generally by Mycoplasma serology
- positive cold agglutination test

Management

- erythromycin/clarithromycin
- tetracyclines such as doxycycline are an alternative



Comparison of Legionella and Mycoplasma pneumonia

External Links

[DermNet NZ](#)

Erythema multiforme

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications:

- COPD with respiratory acidosis pH 7.25-7.35
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD:

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H₂O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H₂O whilst BTS suggest 12-15 cm H₂O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

External Links

[RCP](#)

2008 NIV in COPD guidelines

[British Thoracic Society](#)

2008 NIV guidelines

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

Consequence

- daytime somnolence
- hypertension

SIGN guidelines for the diagnosis and management of patients with OSAHS were published in 2003

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

- sleep studies - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- CPAP is first line for moderate or severe OSAHS
- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- limited evidence to support use of pharmacological agents

External Links

[Clinical Knowledge Summaries](#)

Obstructive sleep apnoea guidelines

Oxygen therapy

The British Thoracic Society published guidelines on emergency oxygen therapy in 2008. The following selected points are taken from the guidelines. Please see the link provided for the full guideline.

In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills. The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets

- acutely ill patients: 94-98%
- patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients

- prior to availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO₂ is normal

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- myocardial infarction and acute coronary syndromes
- stroke
- obstetric emergencies
- anxiety-related hyperventilation

External Links

[British Thoracic Society](#)

2008 Emergency oxygen therapy guidelines

Pleural effusion

Exudate (> 30g/L protein):

- infection: pneumonia, TB, subphrenic abscess
- connective tissue disease: RA, SLE
- neoplasia: lung cancer, mesothelioma, metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Transudate (< 30g/L protein):

- heart failure
- hypoalbuminaemia (liver disease, nephrotic syndrome, malabsorption)
- hypothyroidism
- Meigs' syndrome

Pleural effusion: investigation

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion.

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations.

Pleural aspiration

- as above, ultrasound is recommended to reduce the complication rate
- a 21G needle and 50ml syringe should be used
- fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology

Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. **The BTS recommend using the criteria for borderline cases:**

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
- pleural fluid protein divided by serum protein >0.5
- pleural fluid LDH divided by serum LDH >0.6
- pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Pleural infection

- all patients with a pleural effusion in association with sepsis or a pneumonic illness require diagnostic pleural fluid sampling
- if the fluid is purulent or turbid/cloudy a chest tube should be placed to allow drainage
- if the fluid is clear but the pH is less than 7.2 in patients with suspected pleural infection a chest tube should be placed

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation
- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

External Links

[British Thoracic Society](#)

Pleural effusion guidelines

[Royal College of Physicians](#)

2012 Pleural infection review

Pneumonia: community-acquired

Community acquired pneumonia (CAP) may be caused by the following infectious agents:

- *Streptococcus pneumoniae* (accounts for around 80% of cases)
- *Haemophilus influenzae*
- *Staphylococcus aureus*: commonly after the 'flu
- atypical pneumonias (e.g. Due to *Mycoplasma pneumoniae*)
- viruses

Klebsiella pneumoniae is classically in alcoholics

***Streptococcus pneumoniae* (pneumococcus)** is the most common cause of community-acquired pneumonia

Characteristic features of pneumococcal pneumonia:

- rapid onset
- high fever
- pleuritic chest pain
- herpes labialis

Management

CURB-65 criteria of severe pneumonia

- Confusion (abbreviated mental test score $\leq 8/10$)
- Urea > 7 mmol/L
- Respiratory rate ≥ 30 / min
- BP: systolic ≤ 90 or diastolic ≤ 60 mmHg
- age ≥ 65 years

♦Patients with 3 or more (out of 5) of the above criteria are regarded as having a severe pneumonia.

The British Thoracic Society published guidelines in 2009:

- low or moderate severity CAP: oral amoxicillin. A macrolide should be added for patients admitted to hospital
- high severity CAP: intravenous co-amoxiclav + clarithromycin OR cefuroxime + clarithromycin OR cefotaxime + clarithromycin
- the current BNF has slightly different recommendations for high severity CAP: intravenous benzylpenicillin + clarithromycin OR benzylpenicillin + doxycycline. For 'life-threatening' infections the BNF recommends the same as the BTS guidelines for high-severity CAP

External Links

[British Thoracic Society](#)

2009 CAP guidelines update

[Royal College of Physicians](#)

2012 Community-acquired pneumonia: assessment and treatment

[Royal College of Physicians](#)

2012 Influenza-related pneumonia review

Pneumonia: prognostic factors

The British Thoracic Society recommends that patients should be assessed for the severity of their pneumonia using several core prognostic features (CURB-65 score).

The CURB-65 score is as follows:

Criterion	Marker
C	Confusion (abbreviated mental test score $\leq 8/10$)
U	Urea >7 mmol/L
R	Respiration rate $\geq 30/\text{min}$
B	Blood pressure: systolic ≤ 90 mmHg and/or diastolic ≤ 60 mmHg
65	Aged ≥ 65 years

Patients with a CURB-65 score of **0** should be managed in the community.

Patients with a CURB-65 score of **1** should have their SaO_2 assessed which should be $>92\%$ to be safely managed in the community and a CXR performed. If the CXR shows bilateral/multilobar shadowing hospital admission is advised.

Patients with a CURB-65 score of **2** or more should be managed in hospital as this represents a severe community acquired pneumonia.

Chapter: Respiratory medicine

The CURB-65 score also correlates with an increased risk of mortality at 30 days with patients with a CURB-65 score of 4 approaching a 30% mortality rate at 30 days.

Other factors associated with a poor prognosis include:

- presence of coexisting disease
- hypoxaemia ($pO_2 < 8$ kPa) independent of FiO_2

External Links

[British Thoracic Society](#)

2009 CAP guidelines update

Pneumothorax

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2010. A pneumothorax is termed primary if there is no underlying lung disease and secondary if there is

Primary pneumothorax

Recommendations include:

- if the rim of air is < 2 cm and the patient is not short of breath then discharge should be considered
- otherwise aspiration should be attempted
- if this fails (defined as > 2 cm or still short of breath) then a chest drain should be inserted
- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Secondary pneumothorax

Recommendations include:

- if the patient is > 50 years old and the rim of air is > 2 cm and/or the patient is short of breath then a chest drain should be inserted.
- otherwise aspiration should be attempted if the rim of air is between 1-2cm. If aspiration fails (i.e. pneumothorax is still greater than 1cm) a chest drain should be inserted. All patients should be admitted for at least 24 hours
- if the pneumothorax is less than 1cm then the BTS guidelines suggest giving oxygen and admitting for 24 hours

Chapter: Respiratory medicine

- regarding scuba diving, the BTS guidelines state: *'Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.'*

Iatrogenic pneumothorax

Recommendations include:

- less likelihood of recurrence than spontaneous pneumothorax
- majority will resolve with observation, if treatment is required then aspiration should be used
- ventilated patients need chest drains, as may some patients with COPD

External Links

[British Thoracic Society](#)

Pneumothorax guidelines

[Chest x-ray.com \(incorporating AJR guidance\)](#)

Calculate pneumothorax size

[YouTube](#)

Needle Aspiration of Pneumothorax by the NEJM

Pregnancy: DVT/PE investigation

Guidelines were updated in 2015 by the Royal College of Obstetricians. **Key points include:**

- ECG and chest x-ray should be performed in all patients
- compression duplex Doppler should be performed if the chest x-ray is normal - this may provide indirect evidence of a pulmonary embolism and negate the need for further radiation exposure
- the decision to perform a V/Q or CTPA should be taken at a local level after discussion with the patient and radiologist.

Comparing CTPA to V/Q scanning in pregnancy

CTPA	V/Q scanning
CTPA slightly increases the lifetime risk of maternal breast cancer (increased by up to 13.6%, background risk of 1/200 for study population). Pregnancy makes breast tissue particularly sensitive to the effects of radiation	V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA (1/280,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it is often raised in pregnancy.

External Links

[RCOG](#)

Thromboembolic disease in pregnancy and the puerperium: acute management

Pulmonary arterial hypertension: causes and classification

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise. PAH has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)

- idiopathic*
- familial
- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, HIV**, drugs and toxins, sickle cell disease
- persistent pulmonary hypertension of the newborn

Group 2: Pulmonary hypertension with left heart disease

- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia

- COPD
- interstitial lung disease
- sleep apnoea
- high altitude.

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions

- lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

*previously termed primary pulmonary hypertension

**the mechanism by which HIV infection produces pulmonary hypertension remains unknown.

Pulmonary embolism: investigation

We know from experience that few patients (around 10%) present with the medical student textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism *more* likely?

The PIOPED study¹ in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:

- Tachypnea (respiratory rate >16/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

2012 NICE guidelines

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

If a PE is still suspected a two-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores:

- PE likely - more than 4 points
- PE unlikely - 4 points or less.

♦If a PE is **'likely'** (**more than 4 points**) arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give low-molecular weight heparin until the scan is performed.

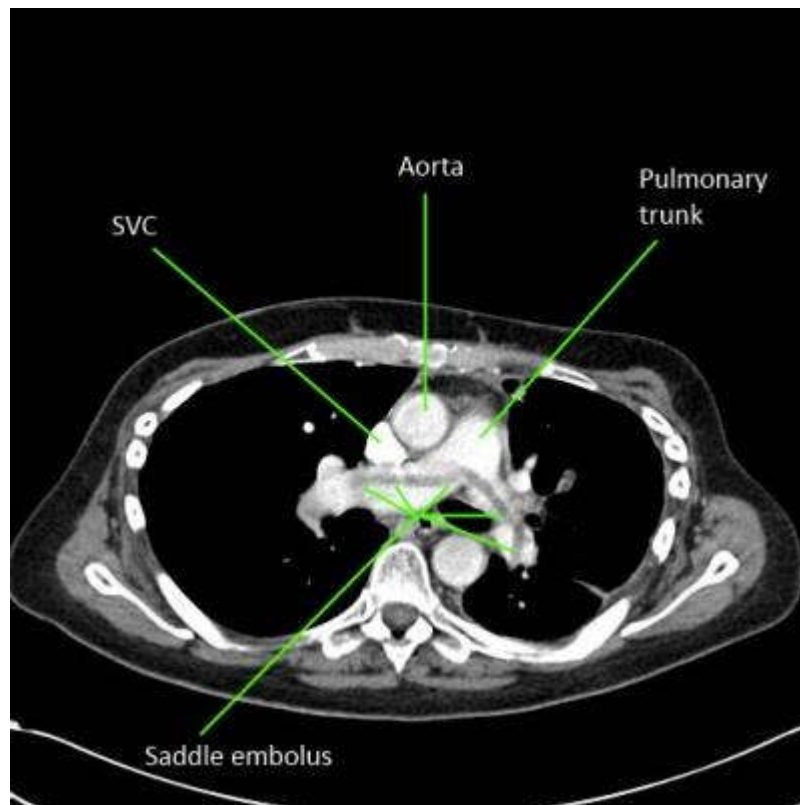
♦If a PE is **'unlikely'** (**4 points or less**) arranged a D-dimer test. If this is positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give low-molecular weight heparin until the scan is performed.

♦If the patient has an allergy to contrast media or renal impairment a V/Q scan should be used instead of a CTPA.

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- computed tomographic pulmonary angiography (CTPA) is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- ventilation-perfusion scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease



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Labelled CTPA showing a large saddle embolus



Further CTPA again showing a saddle embolus

Some other points

D-dimers

- sensitivity = 95-98%, but poor specificity

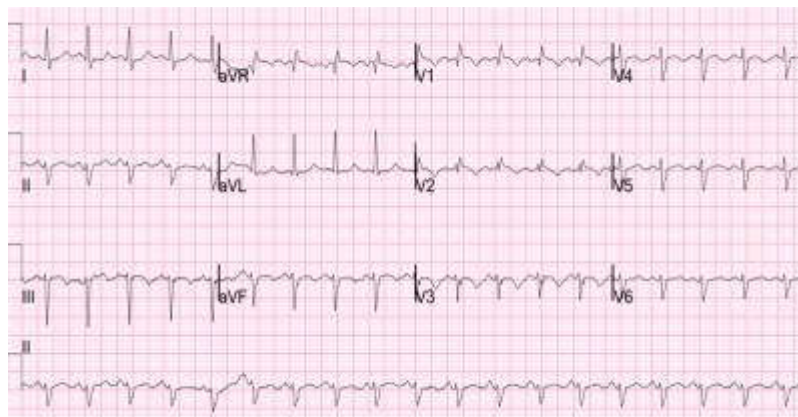
ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen



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ECG from a patient with a PE. Shows a sinus tachycardia and a partial S1Q3T3 - the S wave is not particularly convincing.



© Image used on license from [Dr Smith, University of Minnesota](#)

ECG of a patient with a PE. It shows some of the ECG features that may be associated with PE (sinus tachycardia, S1, T3 and T wave inversion in the precordial leads). Other features such as the left axis deviation are atypical.

V/Q scan

- sensitivity = 98%; specificity = 40% - high negative predictive value, i.e. if normal virtually excludes PE
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed

Pulmonary angiography

- the gold standard
- significant complication rate compared to other investigations

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

External Links

[NICE](#)

2012 Venous thromboembolism guidelines

[Life in the Fast Lane](#)

The ECG in Pulmonary Embolism

Pulmonary embolism: management

♦The NICE guidelines of 2012 provided some clarity on how long patients should be anticoagulated for after a pulmonary embolism (PE). Selected points are listed below.

♦Low molecular weight heparin (LMWH) or fondaparinux should be given initially after a PE is diagnosed. An exception to this is for patients with a massive PE where thrombolysis is being considered. In such a situation unfractionated heparin should be used.

- a vitamin K antagonist (i.e. warfarin) should be given within 24 hours of the diagnosis
- the LMWH or fondaparinux should be continued for at least 5 days or until the international normalised ratio (INR) is 2.0 or above for at least 24 hours, whichever is longer, i.e. LMWH or fondaparinux is given at the same time as warfarin until the INR is in the therapeutic range
- warfarin should be continued for at least 3 months. At 3 months, NICE advise that clinicians should 'assess the risks and benefits of extending treatment'
- NICE advise extending warfarin beyond 3 months for patients with *unprovoked* PE. This essentially means that if there was no obvious cause or provoking factor (surgery, trauma, significant immobility) it may imply the patient has a tendency to thrombosis and should be given treatment longer than the norm of 3 months
- for patients with active cancer NICE recommend using LMWH for 6 months

Thrombolysis

- thrombolysis is now recommended as the first-line treatment for massive PE where there is circulatory failure (e.g. hypotension). Other invasive approaches should be considered where appropriate facilities exist

External Links

[NICE](#)

2012 Venous thromboembolism guidelines

[British Committee for Standards in Haematology](#)

Guidelines on oral anticoagulation (warfarin): fourth edition - 2011 update

[Clinical Knowledge Summaries](#)

Anticoagulation guidelines

[European Society of Cardiology](#)

2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism

Pulmonary eosinophilia

Causes of pulmonary eosinophilia

- Churg-Strauss syndrome
- allergic bronchopulmonary aspergillosis (ABPA)
- Löffler's syndrome
- eosinophilic pneumonia
- hypereosinophilic syndrome
- tropical pulmonary eosinophilia
- drugs: nitrofurantoin, sulphonamides
- less common: Wegener's granulomatosis

Löffler's syndrome

- transient CXR shadowing and blood eosinophilia
- thought to be due to parasites such as *Ascaris lumbricoides* causing an alveolar reaction
- presents with a fever, cough and night sweats which often last for less than 2 weeks.
- generally a self-limiting disease

Tropical pulmonary eosinophilia

- associated with *Wuchereria bancrofti* infection
-

Pulmonary function tests:

Pulmonary function tests can be used to determine whether a respiratory disease is obstructive or restrictive. **The table below summarises the main findings and gives some example conditions:**

Obstructive lung disease	Restrictive lung disease
FEV1 - significantly reduced FVC - reduced or normal FEV1% (FEV1/FVC) - reduced	FEV1 - reduced FVC - significantly reduced FEV1% (FEV1/FVC) - normal or increased
Asthma COPD Bronchiectasis Bronchiolitis obliterans	Pulmonary fibrosis Asbestosis Sarcoidosis Acute respiratory distress syndrome Infant respiratory distress syndrome Kyphoscoliosis Neuromuscular disorders

External Links

lungfunction.net

Tutorial on interpretation of lung function tests

Respiratory acidosis

Respiratory acidosis may be caused by a number of conditions:

- COPD
 - decompensation in other respiratory conditions e.g. life-threatening asthma / pulmonary oedema
 - sedative drugs: benzodiazepines, opiate overdose
-

Respiratory alkalosis

Common causes

- anxiety leading to hyperventilation
- pulmonary embolism
- salicylate poisoning*
- CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- altitude
- pregnancy

*salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis

Respiratory pathogens

The table below lists the more common respiratory pathogens:

Pathogen	Associated condition
Respiratory syncytial virus	Bronchiolitis
Parainfluenza virus	Croup
Rhinovirus	Common cold
Influenza virus	Flu
<i>Streptococcus pneumoniae</i>	The most common cause of community-acquired pneumonia
<i>Haemophilus influenzae</i>	Community-acquired pneumonia Most common cause of bronchiectasis exacerbations Acute epiglottitis
<i>Staphylococcus aureus</i>	Pneumonia, particularly following influenza
<i>Mycoplasma pneumoniae</i>	Atypical pneumonia Flu-like symptoms classically precede a dry cough. Complications include haemolytic anaemia and erythema multiforme
<i>Legionella pneumophila</i>	Atypical pneumonia Classically spread by air-conditioning systems, causes dry cough. Lymphopenia, deranged liver function tests and hyponatraemia may be seen.

Pneumocystis jiroveci	Common cause of pneumonia in HIV patients. Typically patients have few chest signs and develop exertional dyspnoea
<i>Mycobacterium tuberculosis</i>	Causes tuberculosis. A wide range of presentations from asymptomatic to disseminated disease are possible. Cough, night sweats and weight loss may be seen

Rheumatoid arthritis: respiratory manifestations

A variety of respiratory problems may be seen in patients with rheumatoid arthritis:

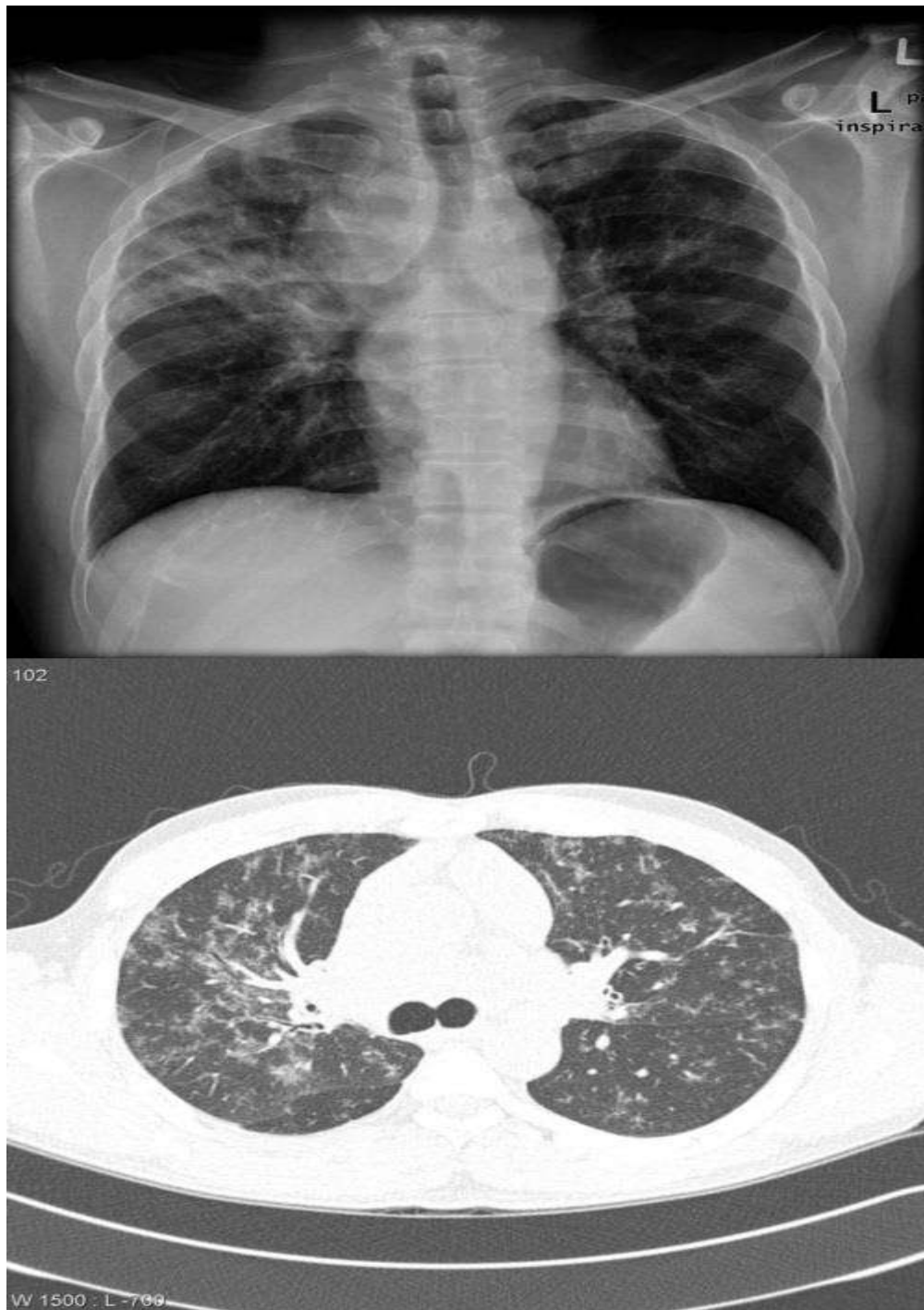
- pulmonary fibrosis
- pleural effusion
- pulmonary nodules
- bronchiolitis obliterans
- complications of drug therapy e.g. methotrexate pneumonitis
- pleurisy
- Caplan's syndrome - massive fibrotic nodules with occupational coal dust exposure
- infection (possibly atypical) secondary to immunosuppression

Sarcoidosis: investigation

There is no one diagnostic test for sarcoidosis and hence diagnosis is still largely clinical. ACE levels have a sensitivity of 60% and specificity of 70% and are therefore not reliable in the diagnosis of sarcoidosis although they may have a role in monitoring disease activity. Routine bloods may show hypercalcaemia (seen in 10% of patients) and a raised ESR

A chest x-ray may show the following changes:

- stage 0 = normal
- stage 1 = bilateral hilar lymphadenopathy (BHL)
- stage 2 = BHL + interstitial infiltrates
- stage 3 = diffuse interstitial infiltrates only
- stage 4 = diffuse fibrosis



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Chest x-ray and CT scan showing **stage 2** sarcoidosis with both bilateral hilar lymphadenopathy + interstitial infiltrates. The reticulonodular opacities are particularly noted in the upper zones. Remember that pulmonary fibrosis (which this case has not yet progressed to) may be divided into conditions which predominately affect the upper zones and those which predominately affect the lower zones - sarcoidosis is one of the former. The CT of the chest demonstrates diffuse areas of nodularity predominantly in a peribronchial distribution with patchy areas of consolidation particularly in the upper lobes. There is some surrounding ground glass opacities. No gross reticular changes to suggest fibrosis.

Other investigations*

- spirometry: may show a restrictive defect
- tissue biopsy: non-caseating granulomas
- gallium-67 scan - not used routinely

*the Kveim test (where part of the spleen from a patient with known sarcoidosis is injected under the skin) is no longer performed due to concerns about cross-infection

Sarcoidosis: management

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent.

Indications for steroids

- patients with chest x-ray stage 2 or 3 disease who have moderate to severe or progressive symptoms. Patients with asymptomatic and stable stage 2 or 3 disease who have only mildly abnormal lung function do not require treatment
 - hypercalcaemia
 - eye, heart or neuro involvement
-

External Links

[Medscape](#)

Management of sarcoidosis.

Silicosis

Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica). It is a risk factor for developing TB (silica is toxic to macrophages).

Occupations at risk of silicosis:

- mining
- slate works
- foundries
- potteries

Features:

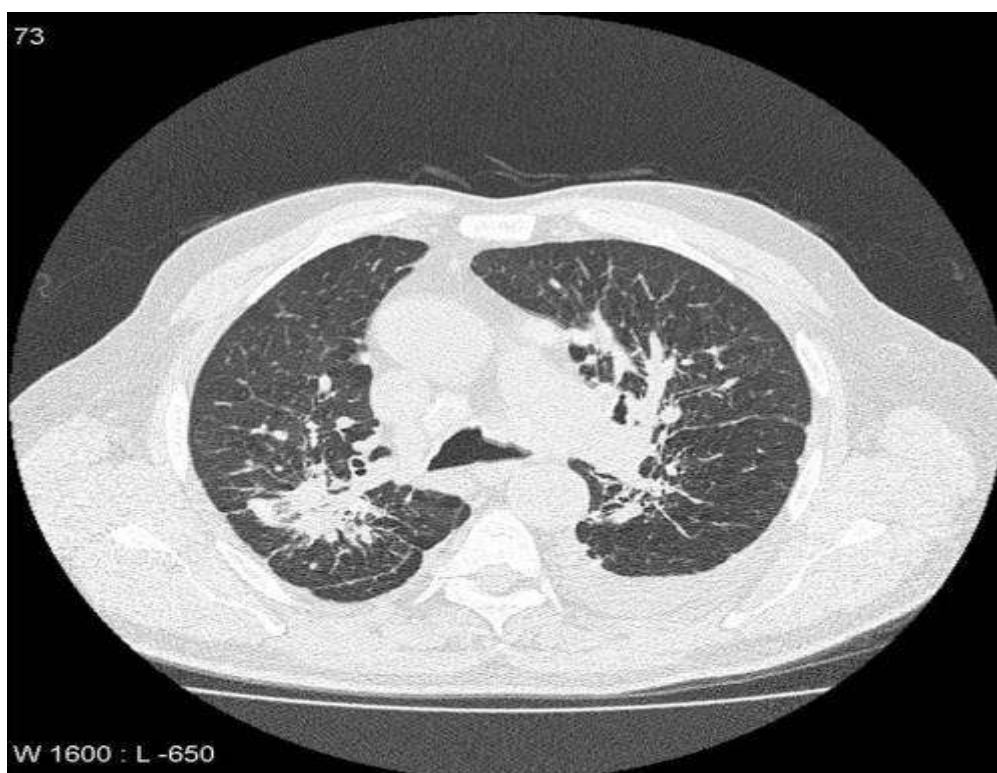
- fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes



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Chest x-ray from a patient with silicosis. Note the bilateral diffuse upper lobe reticular shadowing superimposed with occasional scattered mass like opacities. These features are in keeping with silicosis and progressive massive fibrosis (PMF)



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CT scan from a patient with silicosis showing upper zone predominant mass-like scarring with calcification and volume loss. Hilar and mediastinal lymph node calcification also noted. No cavitary changes are seen. There is a left pleural effusion.

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO

- asthma
- pulmonary haemorrhage (Wegener's, Goodpasture's)
- left-to-right cardiac shunts
- polycythaemia
- hyperkinetic states
- male gender, exercise

Causes of a lower TLCO

- pulmonary fibrosis
- pneumonia
- pulmonary emboli
- pulmonary oedema
- emphysema
- anaemia
- low cardiac output

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO:

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

External Links

[ATS Concise Clinical Review](#)

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

Tuberculosis: drug therapy

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE):

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol (the 2006 NICE guidelines now recommend giving a 'fourth drug' such as ethambutol routinely - previously this was only added if drug-resistant tuberculosis was suspected)

Continuation phase - next 4 months:

- Rifampicin
- Isoniazid

♦The treatment for **latent tuberculosis** is isoniazid alone for 6 months

♦Patients with **meningeal tuberculosis** are treated for a prolonged period (at least 12 months) with the addition of steroids

Directly observed therapy with a three times a week dosing regimen may be indicated in certain groups, including:

- homeless people with active tuberculosis
- patients who are likely to have poor concordance
- all prisoners with active or latent tuberculosis

External Links

[NICE](#)

2016 Tuberculosis guidelines

[Cochrane](#)

Corticosteroids for managing tuberculous meningitis

Tuberculosis: screening

The Mantoux test is the main technique used to screen for latent tuberculosis. In recent years the interferon-gamma blood test has also been introduced. It is used in a number of specific situations such as:

- the Mantoux test is positive or equivocal
- people where a tuberculin test may be falsely negative (see below)

Mantoux test

- 0.1 ml of 1:1,000 purified protein derivative (PPD) injected intradermally
- result read 2-3 days later

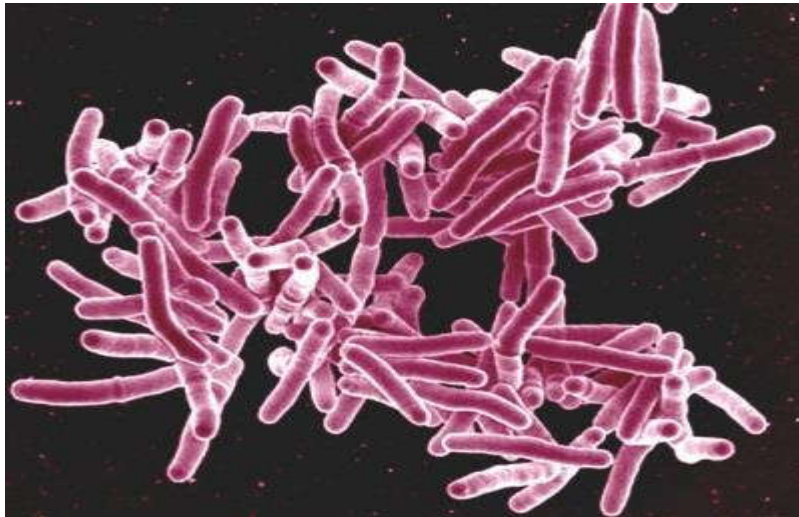
Diameter of induration	Positivity	Interpretation
< 6mm	Negative - no significant hypersensitivity to tuberculin protein	Previously unvaccinated individuals may be given the BCG
6 - 15mm	Positive - hypersensitive to tuberculin protein	Should not be given BCG. May be due to previous TB infection or BCG
> 15mm	Strongly positive - strongly hypersensitive to tuberculin protein	Suggests tuberculosis infection.

False negative tests may be caused by:

- miliary TB
- sarcoidosis
- HIV
- lymphoma
- very young age (e.g. < 6 months)

Heaf test

The Heaf test was previously used in the UK but has been since been discontinued. It involved injection of PPD equivalent to 100,000 units per ml to the skin over the flexor surface of the left forearm. It was then read 3-10 days later.



Scanning electron micrograph of Mycobacterium tuberculosis bacteria, which cause TB.
Credit: NIAID

External Links

[NICE](#)

2016 Tuberculosis guidelines